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EDITORIAL

THE MEDICAL SOCIETY

It is hard for the student in Medicine to understand the Artsman's enthusiasm for student government. Articles in the Varsity on the subject—especially long accounts of proposed changes in the constitutions of the large colleges—fill most of the members of the medical society with amazement, and fill those anxious to see publicity for Medicine with fury. A great deal of the amazement and a great deal of the fury is justified. Equally justified is the amazement, and perhaps the fury with which the Executive of the Medical society views the indifference of the members to the affairs and functions of the society. Recently such a member told us that he did not believe it mattered who was elected president of the society, for things would carry on as before in any case. It might be worthwhile to have two or three very bad executives in order that the members might have a clear demonstration of the importance to them of a smoothly functioning society, and of the importance of their duty to maintain it, watch it, and to take part in its activities.

We have used the noun 'member'. That is the first important fact about the Society. Every undergraduate in the Faculty of Medicine is a member. The function and purpose therefore, is service to the members, and this involves a great many concrete facilities, opportunities, luxuries, and pleasures.

Historically, an undergraduate organization is first formed in order that there may be an orderly transfer of opinion and information between the faculty and the students. This is still a most important business. During the course, we have all benefitted from changes in examination dates arranged by the year president. Similarly, the president of the Society is able to make arrangements for our convenience through the Dean. The Curriculum Committee provides a channel through which students may express their opinions of the course.

The Meds-At Home stands out in every mind as the central social event of the year. Nothing depends so much on its directors as a dance. If you thought the dance was good, then give credit to the dance chairman. If you thought the dance was poor, blame yourself, for you elected him. And be certain, that if a good man is not elected, the dance will be poor. However, be careful about voting for a candidate who promises you a good time at the At-Home. Remember, he cannot control the company you chose.

For those who love sports, the Medical Athletic Society is all important. A very large part of the budget of the Medical Society goes for equipment and other athletic expenses. We need not stress the loss, both in fun and sense of well-being if the athletic representatives were not interested in getting as many medsmen out for sports as can be persuaded or forced to play.

Constitutionally, the Medical Society fosters culture. The most notable of its cultural activities is the Daffydil Night. Since its revival, this has been an evening to look forward to all fall, and to look back on all winter. For the increasing number who take part in the show, either before or behind the scenes, there is a large reward in personal satisfaction that comes with being part of a group enthusiastic about their job. More truly cultural, although perhaps not so popular, are the Medical Arts and Letters club and the Medical Music Association. Both have been warmly supported by the executive. Those who have taken part in their activities have gained by it. Those who have not, would be well advised to plan to be active in one or both next year.

It is our opinion of course, that this Journal is the most important thing which the society gives to its members. Certainly, because of rising prices, it is one of the most expensive of the services. Its future depends on the quality of the staff selected by the executive, and more particularly on the interest of the students.

It has been easy for us to show the importance of the internal activities of the society. Less obvious is the importance of the society as a link between our faculty and the other colleges and Faculties, and the University as a whole. It is a great honour to Medicine that for the last two years the President of the Medical Society has been elected president of the Students Administrative Council. It is the duty of our representatives to the Council to see that Medical

students have the opportunities to benefit from the services of the council, and the information necessary to seize the opportunities. It is our opinion, that, by expressing moderate and reasonable views these represntatives have increased the prestige of the body that sent them, and performed a service for the whole university.

Perhaps the most certain fact about the Society is that it has lots of room for improvement. One cannot attend a single meeting without being struck with this need. Had the Journal a few hundred more dollars, we could spend a few pages listing possible improvements. However, an evolutionary process is taking place, and with the intelligent interest and guidance of the members, the society will grow in stature beyond recognition in the next ten years.

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SOME ASPECTS OF CORONARY ATHEROSCLEROSIS AND ITS SEQUELAE

A. J. KERWIN, M.D.

One hundred and fifty years ago the average expectation of life at birth was between 30 and 35 years. Improvement in nutrition, public health and particularly in the prevention and treatment of bacterial diseases have caused a great decline in mortality in childhood, adolescence and early adult life so that now the life expectancy is between 60 and 65 years and is increasing with each year. It is probable that the ultimate attainable in the light of present knowledge is about 70 years. Any further increase will necessitate a better understanding of the diseases of senescense.

Of these the largest and most important group is arteriosclerosis, particularly when it involves the coronary arteries. Even yet, nothing of fundamenal significance is known about the etiology. Age is important in most cases but an advanced degree of the disease is not unheard of in small children. Similar lesions can be produced experimentally in animals but not under circumstances comparable to those in which the condition occurs in humans.

Pathology: It is unfortunate that the generic term "arteriosclerosis" includes a variety of lesions, more or less distinct in type and location and probably of differing pathogenesis. From the practical standpoint when we speak of arteriosclerosis of the coronaries we mean atherosclerosis which is a subintimal lesion consisting of hyaline thickening, lipoid deposits, calcification and secondary atrophy of the media. In itself coronary sclerosis is of no importance except as (1) it interferes by obstruction with the sole function of the arteries, namely, the supply of sufficient oxygen and nutriment to the muscle and/or (2) forms the starting point of a sudden thrombotic occlusion.

The presence or absence of atherosclerosis or other types of arteriosclerosis elsewhere in the body gives no certain indication of the status of the coronary system. Thickening of the radials is of no value in assessing the coronaries!

While it is essentially a disease of older age group, not rarely it is well developed, anatomically if not clinically, in the twenties and thirties. Contrariwise, the coronary arteries of the aged are occasionally strikingly free from it. The magnitude and distribution of the lesions are highly irregular though usually the main trunks are most involved. Because of the small size of these vessels and the enormous demand on them as conduits (the resting blood flow is estimated to be as much as 500 cc. per min.), small nodular lesions strategically placed may have serious and far-reaching effects.

The location and speed of development of these lesions depend on factors as yet unknown but the influence on the subsequent course of events is all important. In normal hearts there is a limited anastomotic circulation between the main branches of the system; Blumgart and others found the anastomotic channels to be less than 40 micra in diameter. The presence of atherosclerotic nar-

rowing of main vessels appeared to induce the enlargement of collaterals to aid circulation in deficient areas. No such finding was noted in the hearts of elderly individuals with an insignificant amount of coronary disease. Hence age alone was not a factor. However, this compensatory process has its limitations and eventually progressive sclerosis produces serious results.

The term "coronary occlusion" refers either to the atherosclerotic nodules which alone may sooner or later obliterate the lumen or to the superimposed thrombus which finally plugs it. These occlusions which are so often multiple, produce necrosis of muscle either slowly or rapidly. If sufficient myocardium is injured, the efficiency of the heart is impaired. In many, gradual replacement of muscle fibres by inert fibrous tissue takes place but sudden and dramatic changes may occur which we know as infarction.

Infarction, or sudden massive necrosis, is usually the result of thrombosis but in 20 to 30% of cases no recent thrombus can be found and only severe sclerotic narrowing or old organized thrombi are present. The pathogenesis of the infarction is here not so obvious as when a fresh thrombus occurs, but presumably depends on a sudden decrease in the available oxygen supply which was barely sufficient before. Then too, coronary thrombosis is not inevitably followed by infarction and a previously expanded collateral circulation may be adequate to prevent necrosis, at least as an immediate effect.

It is still common practice for the physician to speak of "coronary thrombosis." For the reasons noted above, it is preferable to use the term "cardiac infarction." A mass of dead muscle, however produced, is the fundamental alteration on which the diagnosis (apart from the history) is made through its effect on the white count, sedimentation rate, temperature and electrocardiogram. A clinical diagnosis of coronary thrombosis without infarction in the absence of these effects is seldom clinically justified, though it probably occurs oftener than we suspect. No doubt some cases of angina of sudden onset, where thrombosis has decreased the circulation to a portion of myocardium short of actual necrosis, fall into this category.

We know little about the events in the coronary artery in the early stages of acute occlusion. A number of factors are recognized as favouring intra-vascular thrombosis but none of these can be regarded as of proven significance in coronary thrombosis. Subintimal haemorrhage into atherosclerotic plaques is believed by some observers to be important in initiating thrombosis but this has not been widely accepted as a major cause. The histologic appearance of the intima gives no certain information as to why thrombosis should have occurred at that time. Thrombosis may be superimposed on a large nodule nearly occluding the lumen but often occurs on a plaque where there is no significant obstruction. The deposit forms over an area usually less than 5 mm. in length. The speed with which it develops no doubt varies greatly from case to case. In some this happens very rapidly, if one may judge from the clinical course. In others it is probably a slower process but just how slow it may be it is impossible to say. One sees the occasional patient with repeated attacks of severe anginal pain for several

days before the major event. It is conceivable, though unprovable, that the thrombus is being slowly built up during the period of angina and is then followed by rapid complete occlusion.

In those who survive the acute attack, the necrosis of muscle is followed by inflammation and repair, the dead muscle being broken up, absorbed and replaced with scar tissue. Regeneration of muscle fibres does not take place so far as we know. The ultimate effect on cardiac function will depend on the amount of muscle killed in relation to the efficiency of the remainder. If the latter is good, no detectable decrease in function will result. If there is considerable previous damage, varying degrees of lowered cardiac reserve or even actual failure may ensue. Diminished blood supply to nearby areas which have not undergone necrosis, may become the site of origin of anginal attacks not suffered previous to the infarction. If necrosis is extensive, softening and rupture of the myocardium may take place, usually in the second week after the onset. Occasionally the scarring is so extensive that aneurysm of the ventricular wall gradually develops.

In about two thirds of cases of infarction a thrombus forms on the ventricular wall contiguous to the area of necrosis. This begins about the third day and may reach a considerable size. It may be the point of origin for emboli which, since the left ventricle is most commonly involved, will enter the systemic circulation and produce infarcts of the brain, limbs or viscera. Less often will pulmonary infarcts occur from infarctions of the septum with thrombus on the right ventricular wall. Most pulmonary emboli however, as in other bed-ridden patients, arise from phlebothrombosis of leg veins.

Symptoms and Signs: *Atherosclerosis*—In the absence of angina, or failure, coronary atherosclerosis without infarction produces no symptoms per se and may be impossible to diagnose. When sufficient myocardium is damaged, symptoms of decreased cardiac reserve appear. Breathlessness on exertion is the earliest complaint but if the exertion required to bring this on is considerable, it may be difficult to determine whether it is abnormal. Age, obesity and physical "condition" must be taken into account in the evaluation of dyspnoea. As a standard of comparison it is useful to ask the patient if he is more short of breath than his friends or colleagues under similar circumstances. It must always be remembered that dyspnoea is not necessarily cardiac in origin. Shortness of breath on exertion may progress slowly over years or rapidly over weeks so that eventually slight or no effort is needed to evoke it. By this time signs of failure have almost always become evident. Attacks of acute pulmonary congestion may occur during the night. These are often called "cardiac asthma" but are more properly designated "paroxysmal nocturnal dyspnoea" since they are not bronchial in origin. Consequent upon left ventricular failure and pulmonary congestion, signs of right ventricular failure appear—venous congestion, liver enlargement, ascites and dependent oedema. None of these is of course peculiar to coronary heart disease and they depend only on the inability of the heart to act as a fully efficient pump.

Angina—The vast majority of cases of angina pectoris are due to coronary atherosclerosis. It is generally agreed that myocardial anoxaemia is the basic cause of the distress but angina is a transient symptom while the organic lesions cannot vary in degree from moment to moment. This is of course only one aspect of the problem of the relationship of intermittent symptoms to permanent structural lesions anywhere in the body. In a typical case where substernal distresss comes on while walking uphill, against a cold wind or after a meal it seems easy to explain the pain as due to a temporary discrepancy between the oxygen demand and the supply without need of postulating any sudden change in the calibre of the coronary vessels. This explanation appears inadequate where angina develops at a complete rest or as a result of emotional upset. Coronary spasm is at present an unfashionable explanation but one of the lessons of medical history is that discarded theories have a habit of re-appearing. There is suggestive evidence in animals that irritations of the nerves in coronary artery walls without occlusions can cause pain. The inherent difficulties of the problem in the human make it unlikely that a fully satisfactory explanation will soon be forthcoming.

The typical attack of angina is easily recognized but many cases have features which may result in a missed diagnosis. Pain may be absent and the patient complains only of discomfort, burning, choking or tightness. The location may be anywhere between the jaw and the epigastrium. It may commence in an unusual site and be followed later by substernal distress. Radiation down one arm, usually the left, is common but by no means essential to the diagnosis. If exercise is the precipitating factor, there may be great variation in the amount necessary to produce it from day to day. The intensity of the discomfort differs considerably from patient to patient and is no guide to the seriousness of the underlying disease. It is important to realize that this is a diagnosis based on history alone and it is not vitiated by the absence of clinical or electrocardiographic findings. The direct detection of coronary atherosclerosis itself is beyond the reach of present methods of examination!

Infarction: The background of cardiac infarction is usually the same as that of angina. Whereas angina is a transient though recurrent disturbance, infarction is an irreversible organic change in the myocardium dependent on prolonged local hypoxia. The clinical manifestations are somewhat similar to angina but actual pain is more common, at least in the diagnosed cases, and often more intense. However, thrombotic occlusions both old and recent are not infrequent autopsy findings in cases where a history of the event is entirely lacking. Some apparently have no symptoms whatever; in others angina of sudden onset or a rapid and considerable decrease in exercise tolerance or the sudden appearance of failure may indicate to the wary that infarction has occurred. The differentiation from angina depends partly on the duration of the discomfort which lasts from an hour or so to several days. Dysponea, cyanosis, fall in blood pressure, pulsus alterans, gallop rhythm, arrhythmias and signs of failure may result from the severe myocardial damage. Fever, leucocytosis and increased sedimentation

rate are commonly seen in the first few days and the latter may persist for several weeks. Serial electrocardiograms are of great value in the diagnosis but changes may sometimes be slow to make their appearance and occasionally will be equivocal or absent when the clinical diagnosis seems to rest on secure grounds.

Differential Diagnosis: Where cardiac failure or diminished reserve is the only finding, coronary heart disease must be distinguished from heart disease due to rheumatic fever, syphilis and thyrotoxicosis. The history, physical findings, presence of characteristic murmurs, positive serologic tests and elevated basal metabolism will assist in the differentiation. Space does not permit a detailed discussion of these points. Angina may be confused with spondylitis, effort syndrome, pericarditis, cholelithiasis and many other causes for pain within the same distribution. More urgent is the differentiation of infarction from other acute emergencies. The pain of dissecting aneyrysm is nearly always exceedingly severe, radiates widely to neck, back and limbs and typically reaches its peak of intensity almost at once, whereas the pain of infarction requires much longer to achieve its maximum. Pulmonary embolism sometimes closely simulates infarction but if the embolus is large, dyspnoea will overshadow the pain and acute venous congestion may be evident. Signs of phlebothrombosis of the legs may reward a careful search and reveal the source of the embolus. Haemoptysis is frequently absent and does not appear for a day or more. The importance of the correct diagnosis in perforation of an abdominal viscus or in acute pancreatitis is obvious. In spontaneous pneumothorax dyspnoea is the outstanding symptom and though the pain may be sharp and severe it tends to subside fairly soon. Lobar pneumonia seems to be less common at present but characteristically has chill, fever, pleural pain and cough at the onset. A prolonged anginal attack or "acute coronary insufficiency" may provide difficulty and final decision may have to await changes in the white count, sedimentation rate and electrocardiogram.

Prognosis: Prognosis must always be guarded, having due respect for the patient's morale. Sudden death is most commonly due to coronary disease, probably as a result of ventricular fibrillation or cardiac standstill or more rarely cardiac rupture. Death from progressed cardiac failure is the usual end result of coronary atherosclerosis. Repeated attacks of infaction are not uncommon and the outlook is of course worse with each episode. While one may with reasonable accuracy predict the outcome in a sufficiently large group of patients, as usual, in the individual case the way of the prophet is studded with pitfalls.

Treatment: The ideal approach to therapy in any disease is a direct assault on the cause. However since we are ignorant of the etiology of atherosclerosis and cannot at present halt its progress, we are forced to devote our attention to prevention and treatment of the complications and associated diseases. There is good evidence that diabetes can cause atherosclerosis to appear earlier and in more severe degree than would otherwise be the case. It is reasonable to hope that adequate control of diabetes, aside from its more obvious benefits, will prevent the aggravation of atherosclerosis. Hypertension not only seems to increase the amount of sclerosis but also augments the strain on a poorly nourished

myocardium. Its medical treatment is highly unsatisfactory though rest and sedatives may result in temporary improvement in some cases. In some quarters doubt is beginning to be expressed that thoraco-lumbar sympathectomy will fulfill its earlier promise.

There are general measures which can be applied to these patients whether suffering from decreased exercise tolerance only, actual failure, angina or infarction. Obesity, besides having other deleterious effects, increases the work of the heart and should be corrected. Adequate rest is necessary but the amount advised will vary greatly depending on the individual case. Exercise within the limits which do not produce symptoms probably does no harm and may in fact improve morale and the general physical condition. In the predisposed person too severe restriction of activity is likely to add a cardiac neurosis to the organic lesion and the former is often much more difficult to treat. Even in the least involved case it is best to avoid extremes of exertion since infarction occasionally takes place under these circumstances.

Space does not permit a detailed analysis of the treatment of failure. Suffice it to say that the backbone of therapy is rest, digitalis, sodium restriction and diuretics. Aspiration of fluid from chest or abdomen may give considerable relief from dyspnoea. In some cases of severe venous engorgement, particularly if acute, rapid removal of 500 c.c. of blood from a vein may induce striking though temporary improvement.

Elimination or reduction of factors which produce anginal attacks is possible in most cases though they cannot often be entirely abolished. If a given situation is known from past experience to be likely to cause angina and the stimulus cannot be avoided, the use of a vasodilator before the event will often permit the effort to be carried out without pain. The most useful rapid vasodilator is nitroglycerine, usually in a dose of 0.6 mg. under the tongue. If there are unpleasant side effects from this dose, equally good and less disturbing results can often be obtained from half that amount. Amylnitrite though very rapid in its action is rarely necessary or desirable because of the severe throbbing it produces in the head. Other nitrites are not much used because they are slower to take effect.

The immediate treatment of infarction is directed to the relief of pain. In the experience of most, morphine is the best opiate. Seldom is a dose less than 15 mg. useful and it may need to be repeated in a half hour or sooner. In urgent cases intravenous administration may be advisable. In cases of known sensitivity to morphine and its allies, demerol may be preferred. Complete bed rest is an absolute necessity and the patient should be nursed and fed without effort on his part. Oxygen is indicated for severe dyspnoea or cyanosis. If cardiac failure develops, digitalis should not be withheld. Venesection should be considered where cardiac failure is acute. The period of bed rest is usually from four to six weeks but may be prolonged to several months in severe cases. The subsequent treatment of diminished cardiac reserve, angina and of failure will follow the principles outlined above.

BRONCHIECTASIS

D. N. McLEOD, 4TS

Bronchiectasis is a dilatation of the bronchi, either local or general.

Incidence

Of the chronic pulmonary diseases, bronchiectasis ranks next in frequency to pulmonary tuberculosis. Hedblom (1) and Ochsner (2) claim that its incidence exceeds tuberculosis. Necropsy incidence is about 2.5%. In 400 living cases reported by Perry and King (3) the age incidence of onset was as follows:

less than 10 yrs.—42%
11 - 20 yrs.—27%
21 - 40 yrs.—24%
over 40 yrs.—7%

Thus in this series of cases, 69% of them began in the first two decades of life.

Bacteriology and Pathology

The bacteriology of bronchiectasis is varied, numerous, and mixed. Staphlococci, streptococci, H. influenzae, spirochaetes and fusiform bacilli form the bulk of the organisms usually found present.

Bronchiectasis may be cylindrical (the common form) or saccular. It is usually bilateral, but often unilateral, usually affecting the lower lobes. The left lung is a more common site than the right, with the lower left lobe and the lingula of the upper left lobe being the most common sites.

The bronchus is dilated and filled with pus; the mucosa is hypertrophied and often in folds.

The microscopic picture is that of a chronic abscess. The wall of the bronchus is infiltrated with inflammatory cells, and there is destruction of the musculo-elastic tissues, and the cartilaginous rings except in the so-called "dry bronchiectatics" (Mallory, 4). Sometimes there is a metaplasia of the lining columnar epithelium into a squamous type (Robinson) with loss of bronchial cilia. Often there is a marked obliterating endarteritis with septic thrombosis. From this embolus formation may take place (S).

Pathogenesis

Here, four factors about the disease must be considered (4)

1. Bronchiectasis is rarely a diffuse disease of the bronchial tree, but characteristically involves a group of adjoining bronchi.
2. Bronchiectasis is not usually a progressive disease unless there are repeated atacks of pneumonia.
3. The surrounding lung parenchyma is abnormal.
4. Although bronchiectasis can occur at any age, it is characteristically a disease of youth.

Chronic bronchial infection may be primary and may induce bronchial dilatation by injury to the skeletal structures of the bronchial wall. In stating this the following three theories must be discarded.

(1) Chronic cough was thought by some to have a dilating effect on the bronchi but Andrus (5) has shown that during the cough the pressure is greater in the alveola than in the bronchi, which would tend to constrict them.

(2) The deep inspiration preceding the cough has a bronchodilating effect, but its brief duration, plus the fact that the sudden deep inspiration taken in by a bronchiectatic in sports has never been shown to be a causative agent, condemns this theory.

(3) Pressure of stagnant secretions has also been suggested as a possible factor, but does not explain the factor in the "dry" types of bronchiectasis.

Congenital abnormalities may be factors in the production of bronchiectasis. The condition of congenital cystic disease is rarely a factor. Here the outgrowths or buds of bronchial tissue may produce cysts which become distended with mucinous fluid, if the lumen of the stalk becomes obliterated.

An indirect result of congenital influence is clearly shown by the cases of situs inversus with bronchiectasis of the lower lobe and coincident sinusitis. The contention of a predestination in bronchiectasis by Sauerbruch (6) has no positive proof.

Bronchiectasis may be caused by bronchostenosis which includes:

(1) aspirated foreign bodies.
(2) intrabronchial tumours.
(3) bronchial cicatrics—syphilis, tuberculosis.
(4) enlarged hilar lymph glands—tuberculosis, lymphoblastoma.

Stenosis of major bronchi is frequently associated with progressive ectasia of the subtending divisions. But this again is not always so, as shown by the fact that in chronic asthmatic patients, bronchiectasis is extremely rare. Thus if obstruction is incomplete and expiration is impeded more than inspiration, the alveoli rather than the bronchi tend to dilate and emphysema results. With complete obstruction, atelectasis follows and this is an important factor in the development and chronicity of the infection.

The mechanism by which atelectasis induces bronchial dilation is as follows. With collapse or destruction of large amounts of lung tissue, a potential space is created within the plural cavity, and the negative intraplueral pressure increases to around — 30 mm. Then the diaphragm rises on the affected side, and the neighbouring pulmonary tissue overexpands. This is not enough to compensate for the increased negative pressure, and this negative pressure is transmitted through the solid non-expanded pulmonary tissue to the elastic and expandible bronchial walls. When the traction force is exerted mainly on the medium sized bronchi, the dilatations are cylindrical or fusiform, but when the obstruction is down in the finest bronchioles (which contain no cartilege and little muscle), the dilatations

are likely to be saccular. It is to be noted that when air is periodically introduced into the plueral cavity, and the negative pressure does not develop, pulmonary atelectasis may be maintained for long periods of time without danger of bronchial dilatation.

Some state that even then bronchiectasis will not develop unless the bronchi are simultaneously infected. There is no proof of this in man.

In all reports of series of bronchiectatic case histories, a history of pneumonia, or of an upper respiratory infection, often complicated by pneumonia, such as measles, whooping cough, and influenza has been obtained in over 50% of the patients (Perry and King). Other patients trace the onset of their disease to a tonsillectomy or to an aspiration of a foreign body—both common causes of pneumonitis. It seems that bronchiectasis and sinusitis are two distinct conditions, and that sinusitis is only rarely if ever the cause of bronchiectasis (Goodale 7). Thus five factors have been suggested in the pathogenisis of bronchiectasis.
These are:

(1) Direct bronchial infection
(2) Congenital malformation of the bronchial tree.
(3) Bronchostenosis.
(4) Pulmonary atelectasis
(5) Pneumonitis with its sequel, pulmonary fibrosis.

Thus bronchiectasis is generally a disease of the pulmonary parenchyma. as well as the bronchial tree itself.

Diagnosis

A good history is the most important factor in the clinical diagnosis of bronchiectasis. There is often a typical story whooping cough or measles in childhood from which recovery was slow (9). Sometimes the story dates back to the time the patient had a tonsillectomy. The bronchiectatic then may give a history of a troublesome cough each winter, or of having recurrent attacks of pneumonia or pleurisy. A history of having a persistant chronic cough with the expectoration of foul smelling sputum is the most constant finding. The cough is increased with postural changes (such as bending over), exercises and excitement. The sputum is usually yellowish green in colour and has a foul odour. A history of haemoptysis at one time or another may be found in 50% of the cases. Cyanosis and dyspnea are late manifestations of the disease, while such constitutional symptoms as intermittent fever, fatigue, and loss of weigh are only seen in the more severe cases.

Physical examination may reveal absolutely nothing. However there may be dullness over the effected areas, and moist râles, often post tussic, are the most consistent chest finding. Clubbing of the fingers exists in about 50% of the cases.

A plain X-ray often shows nothing, but pulmonary infiltration and fibrosis may be seen. A bronchoscopic examination should then be made to rule out, or discover any obstructing lesion. A positive lipiodol bronchogram showing the fusiform or saccular dilatations of the bronchial tree clinches the diagnosis. In perform-

ing this, lipiodol is introduced into the bronchial tree and X-rays are taken after the throat and larynx have been successfully anaesthetized with local anaesthetic.

Treatment

1. Surgical:

The only cure for bronchiectasis is by total extirpation of the diseased parts of the lung. Thus lobectomy is the treatment of choice (preferably between the ages of 8-21 years) (10). The mortality rate in unilateral disease is small (2-3%). There has not been a death in a special series of over 100 cases in the Toronto. General Hospital (Janes). The mortality rate for bilateral lobectomies is higher (15-25%) (13).

2. Medical:

In any large number of patients with bronchiectasis, about 50% of them are unsuitable for surgery (Alexander) (11). These patients not suitable to surgery are those with too much or too little bronchiectasis, those with limited cardio-vascular reserve, those with limited pulmonary reserve because of associated emphysema, those with concurrent disease, and those who will not submit themselves to surgery.

The most important management of bronchiectasis is mechanical drainage (12). This is done by the patient leaning over a high bed with the lower half of the body resting on the bed, and the head near the floor. This causes the secretions to run up into the normal parts of the bronchi, which causes a cough reflex and the sputum is coughed up. However if a high bed is not available any kind of an article with a flat top and sufficient height off the floor will suffice. Postural drainage may even be done by standing on the floor and bending over. Drainage after a meal is the best time, for then the internal mucous glands of the body are in action, and it is much easier on the patient. Expectorants and deep breathing also aid in emptying deep thick secretions. Drainage should be carried out as often as necessary, every two hours if required. However most bronchiectatics can get along with two to three drainages per day, after meals. This should be done regularily for a period of ten minutes at the same times each day, for with regular drainage the bronchiectatic gets into the so-called swing of things and feels infinitely better. The position for drainage varies wih certain people depending on whcih bronchus is affected. However the patient can soon tell if there is more drainage while lying verticaly downwards, or deviated to one side.

The bronchiectatic should take care of all respiratory infections. Chemotherapy in the form of penicillin produces no cure for bronchiectasis, but its judicious use can shorten the duration of acute attacks of bronchitis and pneumonitis. Here it should be given intramuscularly because the aerosal penicillin has little or no effect. A helpful hint for those bronchiectatics who suffer from sinusitis is to use sparingly a penicillin nebulizer. This fills the whole respiratory tract with penicillin vapour, and even though the bronchiectasis is not affected, it often relieves the annoying and painful symptoms of sinusitis.

Regularity is the key-word for the medical treatment of bronchiectasis. The perfect treatment should include regular good nutritional meals, regular sleeping hours, and regular postural drainage periods.

Under this treatment, a bronchiectatic should lead a fairly normal life unless infection intervenes. A certain amount of exercise is necessary, but as in the treatment of diabetes, it should approximately be the same amount every day. Smoking, exposure to cold and wetness, exposure to people with respiratory infections, and crowds should be avoided as much as possible.

Two other details are important in the medical treatment of bronchiectasis. The first is that in patients who can afford it, the warm dry climates of Arizona and other places are often beneficial. This is often scoffed at, but many bronchiectatics have gone to these areas, made their home there, and have felt much better. The second point is that psychotherapy is very important in the treatment of bronchiectasis. The doctor must realize that he is dealing with a patient who is the so-called "social outcast". The patient unless he keeps himself well drained goes around coughing, bring up foul looking sputum, has foul breath, cannot take part in sports, and who is often either not invited or afraid to go to parties because of the cough and foul breath. Thus the doctor should have a general idea about what the patient with bronchiectasis has to put up with and thus help him accordingly. In some cases the bronchiectatic comes to see the doctor, not because of the cough and sputum, but because of a slight depression due to not being able to carry on either economically or socially. To remedy this the physician should realize the problems, give reassurance, and explain to the patient his limitations.

3. Prophylatic (10):
   (1) Early diagnosis of pneumonia and upper respiratory infections in childhood with adequate follow-up.
   (2) Re-expansion of atelectases that persist after pneumonia and acute respiratory infection, especially in children.
   (3) The early recognition of foreign bodies and their removal is imperative.
   (4) Deep breathing exercises for the affected side immediately after the acute phase of pneumonia has subsided.

Complications

1. Recurrent bronchopneumonia.
2. Brain abscess.
3. Corpulmonale.
4. Empyema.
5. Pyoneumothorax.
6. Lung abscess.
7. Amyloid disease.

} Least Common

Prognosis

The prognosis seems to depend on the effectiveness of the treatment, and whether the disease is progressive or not. However bronchiectasis is not pro-

gressive providing there are no recurrent attacks of bronchopneumonia (Mallory (4), Coope (9). This can be proven by repeated lipiodol bronchograms.

If however, the individual has a vast lesion so that there can be no surgical intervention, the prognosis is practically hopeless.

If the lesion is amenable to surgery, the prognosis with surgical intervention is excellent. If the lesion is to small for surgical intervention, or if surgery is not performed for some reason in a moderate case of bronchiectasis, the prognosis is fairly good under strict medical treatment, but fairly bad in a patient who is careless in following the routine.

REFERENCES

1. Hedblom, C. A.: Bronchiectasis—Lewis Practice of Surgery W. F. Prior Co., Hagerstown, Vol. 5.
2. Ochsner, A., Bronchiectasis—Am. J. Med. Sc. 179, 338, 1930.
3. Perry, K. M. A. and King, D. S., Bronchiectasis—Am. Rev. Tuberc. 41, 531, 1940.
4. Mallory, T. B., Pathogenesis of Bronchiectasis—New Eng. J. of Med. 237: 795-798, 1947.
5. Andrus, P. M.: Bronchiectasis—Am. Rev. Tuberc. 36: 46-81, 1937.
6. Sauerbruch, F. Bronchiektasin—Arch. f. klin. CHIR 180: 312-320, 1934.
7. Goodale, R. L.: Analysis of 75 cases on bronchiectasis from view-point of sinus infection—Ann. Otol., Rhin. and Laryng. 47: 347, 1938.
8. Boyd, W. Text Book of Pathology—Lea and Febiger, Philadelphia 4th Ed.
9. Coope, R.: Disease of the chest—Livingstone, Edinburgh 1st Ed.
10. Scarlet, E. P.: Bronchiectasis—Can. M.A.J. 54: 275-283, 1946.
11. Alexander J.: Roles of Medicine and Surgery in the Management of Bronchiectasis—Am. Int. Med. 21: 565-579, 1944.
12. Badger, F. L.: Bronchiectasis—New Eng. J. of Med. 237: 937-940 1947.
13. Strieder, J. W.: Surgical Aspects of Bronchiectasis—New Eng. J. of Med. 283: 109-112, 1948.

INTERNAL DERANGEMENTS OF THE KNEE JOINT

HAROLD M. M. TOVELL, B.A., M.D.

The purpose of this article is to discuss internal derangements of the knee joint and to present some of the more important anatomical and therapeutic principles involved in arriving at a reasonable diagnosis and successful management of an injured knee.

Anatomy of the Knee Joint

The anatomy of the knee joint will be discussed under the following headings:

1. Movements of the knee joint.
2. Collateral ligaments.
3. Cruciate ligaments.
4. Semilunar cartilages.
5. Synovial Fat Pads.
6. Muscles acting on the knee joint.

1. *Movements of the knee joint*

The knee joint is a modified hinge joint in that besides flexion and extension, there is a small degree of internal rotation of the femur during the last few degrees of extension. Though there has been much disagreement regarding the precise function and mode of action of the ligaments and menisci of the knee joint, recent studies (1. 2. 4.) on over 100 fresh specimens appear to be conclusive.

Commencing with the hyperextended knee, both collateral ligaments are taught with the anterior aspents of the menisci snugly held between the femur and the tibia. During the initial degrees of flexion, the femur undergoes a slight lateral rotation, the medial condyle hinging on the tibial collateral and posterior cruciate ligaments. Further flexion produces a relaxation of the fibular collateral ligament which is maintained even in full flexion where it assists in stabilizing the joint and preventing rotation.

The tibial collateral ligament remains taut throughout but the area of tension shifts from its full width in extension to its anterior portion in full flexion. In this latter position it also stabilizes the joint and prevents rotation.

Both cruciate ligaments remain taut throughout and tighten on complete flexion (1, 2). Disagreement on this point is based on the assumption that there is a small amount of normal adduction and rotation in flexion. (4).

The menisci, being pushed by the femoral condyles, slide backwards in flexion and forwards in extension, much as a watermelon seed is propelled when squeezed between the fingers and thumb. The medial meniscus moves only a few millimetres while the lateral meniscus slides through almost 1 cm. The posterior aspects of both menisci are snugly held between the femur and tibia in full flexion.

Small amounts of accessory, non-functional movements occur in the normal

knee that should be appreciated lest they be mistaken for an abnormality. They vary among individuals and comparison with the normal knee should always be made. They are (1) Abduction-adduction rocking of the tibia on the femur as noted when testing the collateral ligaments, and (2) antero-posterior mobility of the tibia as noted when performing the drawer sign for testing the cruciate ligaments.

2. *Collateral Ligament*

(a) Fibular

The ligament is round and cord-like extending from the lateral epicondyle to the margins of the head of the fibula in front of its apex. It is conveniently considered as the upward extension of the Peroneus Longus muscle and hence directed postero-inferiorly. It divides the biceps tendon and is separated from it by a bursa. Separating the tendon from the joint is the Popliteus tendon in its synovial sheath. The ligament is not adherent to the lateral meniscus.

(b) Tibial

The ligament is triangular in shape and attached above to the medial epicondyle of the femur and below to the tibia at two points which are (1) posteriorly and immediately below the meniscus and above the Semimembranosus tendon, and (2) anteriorly on the medial surface of the tibia, and about 4.5 cms. below the articular margin immediately behind the pes anserinus. The ligament is separated from the tibia by areolar tissue, the inferior genicular vessels and nerve and a variable number (1-3) of bursae (3). The anterior portion of the ligament is separated from the meniscus by the joint capsule and frequently a bursa. Posteriorly, the fibres are indistinguishable from the capsule and accordingly may be considered attached to the medical meniscus. The posterior oblique fibres of the ligament are relaxed in flexion while the anterior parallel fibres remain continuously taut.

3. *Cruciate Ligaments*

The anterior cruciate ligament is attached to the front of the tibial spine and passes outwards and backwards to the intercondylar surface of the lateral femoral condyle. The posterior cruciate ligament passes from the back of the tibial spine forwards and inwards to the front of the intercondylar surface of the medial femoral condyle. Both ligaments tighten on flexion and prevent excessive antero-posterior gliding of the femur on the tibia during all phases of joint movement.

4. *Semilunar Cartilages*

The round lateral meniscus is smaller in diameter being thicker at the periphery and usually wider than the medial meniscus. It is firmly attached to bone in the intercondylar area of the tibia and to the anterior and posterior cruciate ligaments in front and behind respectively by strong fibrous tissue. The

fibrous capsule blends with its periphery and the Popliteal tendon separates it from the fibular collateral ligament.

The "C" shaped medial meniscus is larger in diameter, thinner at the periphery and has no attachment to the cruciate liagments. Its bony attachments are similar to the lateral meniscus. It is attached to the joint capsule at the periphery and frequently a bursa lies between it and the medial collateral liagment.

There appears to be no definite anatomical reason to explain the higher incidence of injury to the medial meniscus (1, 8), though the greater mobility of the lateral cartilage has been suggested. The reason appears to be mechanical because unless the pressure holding the menisci between the condyles is steadily maintained, they may be caught between opposing surfaces and be subject to crushing injuries that split or loosen them from their attachments. Once the tear has occurred it rarely heals since the structures are avascular. Should the tear occur at the periphery where the blood supply is slight healing may take place.

The menisci cushion hyperextension and hyperflexion (not a blow from above or below because condylar articular contact between femur and tibia is always present). They fill an otherwise dead space at the periphery of the joint, and prevent the capsule and synovia from becoming pinched between the femoral and tibial condyles. They also afford a sliding surface to the delicate articular cartilage during the rotation of the femur.

5. *Synovial Fat Pads*

Like the Ligamentum teres of the head of the femur, the Ligamentum mucosum is a triangular synovial fold filled with fat and somewhat flattened. Its apex is attached to the anterior point of the intercondylar notch of the femur. Its base is opened and extends from the lower border of the patella to the anterior intercondylar area of the tibia. The sides are free and prolonged into fringes called alar folds. The infrapatellar pad of fat fills these horizontally lying folds and should they become elongated they may be pinched between the articulating surfaces.

6. *Muscles Acting on the Knee Joint*

To enumerate the muscles acting on the knee joint would be foolish as they are well listed in the standard textbooks. Is is to be noted, however, that the surrounding muscles of the knee joint form the first line of defence in stabilizing the joint and the ligaments, though stabilizing the joint in extreme flexion and extension (two unusual positions), form the second line of defence, serving as "buffers" against any abnormal displacement. The liagaments have a rich sensory innervation and should they be called into play unexpectedly, strong impulses are transmitted via a reflex arc to the effector muscles that maintain the smooth co-ordinated motion of the joint. Should the ligaments, however, be stretched beyond their capacity abnormally strong impulses arise that show themselves clinically in muscle spasm that follows intraarticular injuries to the knee joint. The spasm is particularly selective for the extensor group of muscles, though the flexor group is affected to a lesser degree. The spasm soon gives way to a hypotonia

that is palpable within two hours and, if sufficiently severe, to a visible atrophy within five days (5). It is interesting to note that no spasm or hypotonia occurs in a traumatized knee joint of a dog that has previously been denervated (7).

The Quadriceps femoris muscle then is of the highest clinical importance for should it be weakened the joint stability will be diminished and further articular damage will result.

Examination of The Injuried Knee Joint

Of primary importance in establishing a proper diagnosis of an injured knee joint is an accurate and detailed history of the injury. Points related and subsequent to the injury must be sought out. The type of injury, position of the knee or leg at the time of injury, site of pain and tenderness, ability to straighten leg, and to bear weight following the injury, any swelling and its time of onset, any abnormal sensations or instability are some of the points to be ascertained.

The patient should be stripped from the waist down so that the thighs may be compared for contour and palpated for difference in tone in the relaxed and contracted state. If there is no palpable hypotonia or visible atrophy within an appreciable time, the lesion is probably not in the joint but outside it.

The patellar tap sign will reveal any abnormal collection of fluid. If the fluid has collected within a matter of a few hours, it is probably a haemarthrosis that has to be explained and not a traumatic synovitis. Traumatic synovial fluid collects in the knee joint over a period of days or weeks, not over a period of a few hours.

Palpation for tender areas will often suggest loose bodies, tears of the collateral ligaments and cartilage lesions.

Ability to fully extend the knee is of value in arriving at a diagnosis. The patient should be seated, and the heal of the extended leg resting on the examiner's knee. When the patient is told to relax his knee, the joint will either remain flexed or go into extension. Persistent flexion is due either to mechanical blocking or muscle spasm. If it is the latter, it is probably due to fluid in the joint in which case, if the knee is slowly pushed into extension it will rebound into flexion. A normal knee never rebounds. If the knee cannot be pushed into extension, either because of pain or mechanical blocking, there is probably an internal deragement of the joint.

Abnormal mobility should be tested, noting the extent of accessory movement in the normal knee. Frequently however, the "drawer sign" for testing the integrity of the cruciate ligaments is unreliable and inconclusive (5, 6, 7).

Certain injuries demand X-ray studies, especially those in which torn ligaments and loose bodies are suspected. Arthrograms are only of value if done by those who are skilled and experienced.

Local injection of 1% Novocain is of frequent diagnostic aid in establishing extra-articular ad capsular injuries. Aspiration of joint fluid is both a diagnostic and therapeutic procedure

Diagnosis and Treatment in General of Internal Derangements of the Knee Joint

The diagnosis of any internal derangement of the knee joint should be based on the following:

1. History of injury
2. Hypotonia with or without atrophy
3. Fluid in the joint
4. Active or passive inability to hyperextend the knee joint.

Whatever the lesion may be, if this much is observed, a great deal has been gained towards arriving at a reasonable diagnosis. If an accurate diagnosis is impossible as is often the case, at least sound therapy directed towards the re-establishment of muscle tone may be instituted.

Some surgeons find it impossible to make a detailed diagnosis and go no further than to make one of the internal derangement of the knee (I.D.K.). This is somewhat analagous to a diagnosis of an "acute abdomen" but is understandable when considering a series of over 400 cases of injured knee joints that were explored. Only 30% showed medial meniscus lesions alone and 70% showed from two to eleven other types of intra-articular lesions (5). Comparable figures have been quoted by others (7, 8.)

For this reason some surgeons prefer a large parapatellar incision as opposed to a small peephole incision, when operating on an internally deranged knee joint. It would appear best to make as careful a diagnosis as possible but also to make as thorough an exploration as can be done without impairing joint function.

Redevelopment of the quadricep muscles for 5 minutes hourly every day is the most important treatment in knee joint injuries. Failure in this will result in an unstable joint that will produce further damage and account for the frequent complaints of a knee "giving way". It is not sufficient to tell the patient to lift a straight leg. He must be shown how to shove back his knee (hyperextend) and to maintain the contraction while raising the leg off the bed. At first the leg is raised against gravity and later weights may be substituted according to the Delorme method.

Frequently there is a post-operative mental alienation to straight leg raising exercises that has to be overcome. This is partially accomplished by instructing the patient to practice active exercises prior to operation and to resume as soon as postoperative conditions permit. The only contraindications to active quadricep exercises are recurrent haemorrhage, open wounds and possible infection.

Early walking has been advocated but this depends on the type of lesion and the condition of the extensor muscles. In properly selected cases there appears to be no delay in convalescence or further disability (9).

Internal Derangements of the Knee Joint

Sprain and Rupture of the Tibial Collateral Ligament

The ligament may be partially or completely torn by an abduction strain of the extended knee. The injury is rare by itself and often occurs with a ruptured medial meniscus.

Local tenderness, swelling, ecchymosis and effusion are accompanied by excessive lateral mobility.

The joint must be immobilized for two months with the valgus deviation corrected and the knee in almost complete extension. Operation is indicated if the ligament is lying within the joint and union fails to occur. Residual pain over the sight of the lesion may suggest ossification of the periosteal attachment of the ligament on the femur—the so called Pellegrini Steida's disease.

Sprain or Rupture of the Fibular Collateral Ligament

Such a lesion is uncommon. When present it may be associated with a torn medial meniscus of the opposite leg as in a bumper injury. The styloid process of the fibula may be avulsed.

Treatment is similar to that for tibial collateral ligament tears, the knee being put into a corrected valgus position and quadricep exercises commenced. Operative procedures are seldom necessary as the lateral side of the joint is well protected by the biceps tendon and the ilio tibial band.

Rupture of the Anterior Cruciate Ligament

Sprains or ruptures may be produced by abduction or hyperextension strains and in both instances there is an associated sprain or rupture of the tibial collateral ligament. A depressed fracture of the lateral tibial tuberosity may occur in abduction injuries.

The abnormal forward mobility of the tibia (Drawer Sign) is the characteristic clinical sign.

Rupture of the Posterior Cruciate Ligament

A violent blow driving the tibia posteriorly on the femur into the popliteal space produces an avulsion of the ligament and often a posterior dislocation of the joint. Clinically there is excessive backward mobility of the tibia.

Treatment of Torn Cruciate Ligaments

The joint must be immobilized sufficiently long, usually two to three months, to permit union of the torn fibres. Residual laxity is minor and of no consequence providing the normal extensor power has been controlled. Many operations have been devised for repair of cruciate ligaments but the results have been no better than those where redevelopment of the quadriceps was the main treatment. In Murray's series of over 400 cases operated on for I.D.K. 9% were associated with an unexpected torn cruciate ligament that showed no functional disability.

Injuries to the Semilunar Cartilages

The mechanism of injury to the menisci is similar in all cases. The medial meniscus is usually torn by an abduction and external rotating strain of the partially flexed knee with weight on the leg as would occur in a sudden pivotting movement. In the case of the lateral meniscus there is an adduction and internal rotating strain on a partially flexed and weight-bearing joint. Rotation is only

possible when the knee is flexed and weight bearing must be present at the moment of injury to give the grinding and splitting force. It is evident that a meniscus lesion is an occupational hazzard and that the medial meniscus will be affected more frequently.
occurred:

Cartilage tears are classified according to whether or not locking has

(1) The bucket handle and peripheral attachment tears. A large fragment or the entire cartilage is displaced medially to produce a mechanical obstruction.
(2) Posterior horn and tears of the central free margin where only a small pedunculated fragment is present.

The diagnostic features of a meniscus tear are a sudden twisting effort of a partially flexed knee resulting in a localized pain either with or without locking of the joint. Effusion may or may not be present. Often the history is characteristically vague and McMurray's sign is of the greatest value in such cases where other signs are wanting.

The treatment of menisci lesions fall into three groups:

(1) Knees that are locked
(2) Those with a first injury
(3) Those with a recurrent injury

The locked knee joint should be reduced under an anaesthetic if possible, not only because it produces the necessary muscular relaxation but also, since failure is otherwise possible, the patients protective reflexes are inhibited and further joint damage is minimized. The hip and knee are flexed to a right angle and the tibia is gently rotated before it is loosely abducted and adducted on the femur. The knee is then suddenly brought into extension to squeeze the meniscus to the periphery of the joint where it remains as long as the extended position is maintained. The knee is then maintained in extension for four weeks and the patient is encouraged to walk gradually.

Operation on initial lesions to the semilunar cartilages will depend on the patients' occupation, age, site of tear and the condition of the extensor muscles. Conservative therapy in doubtful cases is wise so long as the quadriceps is not neglected, but if the symptomatology is at all suggestive, then it is wise to remove the cartilage and prevent further articular damage.

Recurrent injuries may or may not be diagnosed with certainty. If the history and clinical findings are suggestive then a small incision and removal of the cartilage is indicated. Uncertainty however is more often present and in such cases a large parapatellar incision is indicated and a complete exploration of the joint made.

Cyst of the Semilunar Cartilages

Cysts of the semilunar cartilage are occasionally seen. They are probably the result of a direct or indirect contusion producing a mucoid degeneration of the

cartilage. The cyst is bony hard and easily palpable usually at the middle third of the lateral cartilage deep to the fibular collateral ligament.

Persistent pain with no locking or "giving way" at the knee is characteristic. At operation the cyst and cartilage should be removed.

Loose Bodies in the Knee Joint

So-called joint "mice" may arise from three sources:

(1) Chondrification of the synovial membrane with the formation of loose bodies that frequently remain attached to the synovia.

(2) Osteoarthritis with detachment or fracture of marginal osteophytes from the patella, femoral condyles or tibial tuberosities.

(3) Osteochondritis dissecans (see below). [Recurrent pain, swelling and locking of the joint comprise the chief symptoms. Contrast X-ray studies are frequently of value and at operation an exploratory incision is preferable.]

Osteochondritis Dissecans

This is a condition of young people where a fragment of bone and its overlying cartilage sequestrates from a point, usually the lateral aspect of the medial femoral condyle. The etiology is uncertain though the necrosis that is seen has been attributed to an embolic condition interfering with the vascular supply of the bone deep to the articular cartilage. Trauma alone could account for the condition as the milder lesions are frequently associated with an abnormal meniscus, fat tabs and loose bodies. In such cases "chondrosis" would seem a better term. When a portion of the articular cartliage becomes separated, it forms one type of loose body .

Symptoms of vague discomfort soon give way to transitory locking and swelling of the joint. Diagnosis is aided by X-ray studies and treatment is surgical where the softened cartilage and any loose bone is gently curetted.

Lesions of the Fat Pads

A fringe of synovial membrane filled with fat is occasionally pinched between the femoral and tibial articular surfaces. The trauma may be sudden or gradually progressive in onset. In each case there is haemorrhage and laceration with subsequent organization and fibrous tissue formation.

Repeated injuries produce a thick, leathery, tough synovial membrane, with or without adhesions which give rise to the complaints of frequent joint pain and limitation of movement. The diagnosis can only be made at operation where the fat pad is partially or completely removed.

Summary

The more commonly seen internal derangements of the knee joint have been mentioned. Synovial infections, fractures, patellar and bursal lesions have not been discussed though they should be considered whenever confronted with a knee

joint problem. The anatomy of the knee joint has been reviewed and the principles of diagnosing and treating an internal derangement of an injured knee have been given.

REFERENCES

1. Brantigan, O. C. and Voschell, A. F. Journal of Bone and Joint Surgery 23: 44
2. idem Journal of Bone and Joint Surgery 25: 121
3. idem Journal of Bone and Joint Surgery 26: 793
4. Abbott, Le C. et al Journal of Bone and Joint Surgery 26: 503
5. Murray, C. R. New England Journal of Medicine 236: 265
6. idem American Journal of Surgery 55: 262
7. Darrach, Wm. Annals of Surgery 102: 109
8. MacKenzie, D. W. and MacFarlane, J. A. Can. Medical Assoc. Journal 49: 472
9. Jaeckle, R. F. Archives of Surgery 50: 271

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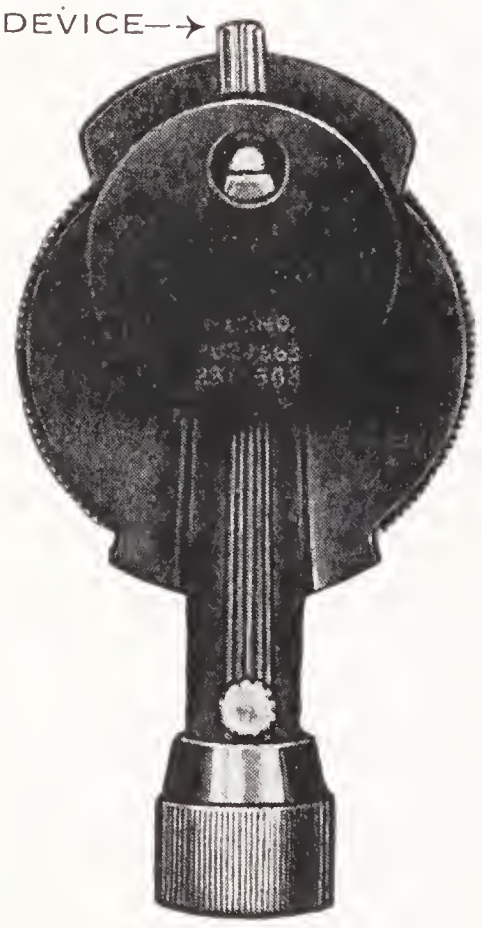

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Feature

R. K. GRAHAM, Editor

THE GRADUATING YEAR—1948

In preparing the statistics which will be presented below a Questionnaire was distributed to members of the graduating year. Although the response was not obtained from every member, nevertheless sufficient responded so that a reasonably good overall picture of the year could be visualized.

With respect to age as of June 1st, the following figures were obtained and compared with last years:

Age in years	'48	'47	Age in years	'48	'47
20	0	0	24	10%	24%
21	1%	5%	25	4%	7%
22	44%	29%	26	2%	5%
23	32%	26%	Over 26	2%	4%

It can be seen that there is a reasonably wide range in ages which on the whole corresponds to the previous year's spread in age. There are 21 girls in the year or 16% of the class which represents an increase of almost a third over our predecessors and probably accounts for our broader outlook.

With respect to outside interests and hobbies it is interesting that in this year as in the class of '47 music in all its varied forms was very much more popular than its runners-up, photography and literature. Next in order came athletics and all forms of sports, camping, and art, together with politics, hunting, church work and model building. As can be seen the list is varied and indicates a wide range of interests and a high level of social responsibility.

One of the important questions in a medical students training is that of post-graduate training and specializing. To properly assess this problem two questions were asked; "How many years would you like to spend in post-graduate training" and "How many years do you think you can spend in post-graduate training." As might be expected the figures are not in any way closely related and indicate that the students wish more training than they will actually receive.

No. of years desired in post-grad. training	% of class	No. of years expected in post-graduate training	% of class
0	0	0	0
1	1.6%	1	27%
2	20%	2	27%
3	32%	3	23%
4	24%	4	10%
5	22%	5	10%
More	.4%	More	3%

The students were asked in which branch of medicine they would like to specialize. At present the sentiment of the class is:

General Practice	—27%
Obstetrics and Gynaecology	—20%
Pediatrics	— 8%
Surgery	—20%
Internal Medicine	—12%
Psychiatry	—10%
Anaesthesia	— .8%
Radiology	— .8%
Neurology	— .8%
Cardiology	— .8%
Ophthalmology	— .8%
Undecided	—17%

The subjects most enjoyed by the students were:

Medicine
Obstetrics and Gynaecology
Surgery
Pathology

At the opposite end of the line was for the second time in two years Hygiene and Preventative Medicine.

On the whole the sort of fellow going through medicine this year is not so very different than the chap or girl who went through last year. During his course his sentiments were divided between Medicine, Obstetrics and Gynaecology, and Surgery, so he will attempt to specialize in all three and become a general practitioner. He will not be bored for he has plenty of outside interests both as skills and as hobbies. And just as in the preceding generations of physicians he will take his prominent place in society.

Abstracts

E. R. YENDT, Editor

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Interosseous Wiring in the Treatment of Fractures of the Mandible. By Stuart Gordon. Archives of Surgery. 55:660 (Dec.) 1947.

The author feels that this treatment of suitable fractures of the mandible has not been used as extensively as its value justifies.

He classes as "suitable" fractures:

(1) Fractures in the edentulous mandible.
(2) Those having an edentulous posterior fragment.
(3) Multiple fractures (to stabilize the main fracture).
(4) Bilateral fractures through the mental foraminae with downward displacement of the central fragment.
(5) Gun wounds.
(6) Fractures at the base of the mandibular condyle, with displacement.

The incision is made below the line of the mandible, the fracture cleanly reduced, and two holes are made in the opposing ends of each fragment. An upper and a lower wire are then inserted, the lower passing under the mandible in a figure-eight manner, to prevent displacement. In nearly all these cases the interosseous wiring was combined with intraoral fixation, to keep movement at the fracture line at a minimum. Post-operative chemotherapy was employed in nearly all cases.

In a series of 46 patients with 48 fractures 1 case of osteomyelitis, one severe and eight mild soft tissue infections, three cases of non-union, and two of sinus due to wire developed. The soft tissue infection was readily controlled by drainage, the non-union by bone graft (using cancellous bone chips), and the sinus formation by removal of the wire.

The author states that interosseous wiring insures obtaining and maintaining a more accurate reduction than any other applicable method. He feels that its use is not contraindicated in compound fractures free of clinical evidence of infection, but should never be used when acute infection is present.

P. G. A.

Influence of Endocrines on Metastases from Prostatic Cancer: C. A. Wattenberg, J. Urol. *58*, 378-383 (1947).

Metastases from carcinoma of the prostate to pelvis, sacrum and lumbar spine are common and cause pain and suffering to the patient. Before the work of Charles Huggins, treatment of secondary metastases was palliative X-ray and succeeded in relieving pain in only half of the cases. Increasing doses of morphine were also required.

In 1941, Huggins first showed the beneficial effects of bilateral orchidectomy and the administration of estrogens as palliation if not cure. Estrogens have been known to produce skeletal changes. Several workers observed hyperossification of bones and hypercalcemia after estrogen administration to animals. Animals under theelin therapy showed faster fracture recovery than similar animals without theelin. Conolly has given estrogen hormones to patients with fractures and has concluded that in older people there is a tendancy to faster callus formation if theelin is administered. These findings of hyperossification fit in well with the results found in the skeletal metastases of prostatic carcinomas after bilateral orchidectomy and diethylstilbestrol therapy. The estrogen causes palliation in two ways: by direct regression of the tumor cells, and by hyperossification about the regressed tumor cells.

Side effects of stilbesterol were noted during treatment. These included breast changes, dependant edema, liver reactions, and changes in the primary carcinoma. The breasts undergo proliferation of the duct epithelium. The edema is due to decreased renal excretion of sodium and chloride ions with consequent fluid retention and reduction in urine volume. There are toxic changes in the liver, increased glycogen storage, and hypertrophy and dilatation of the bile ducts. In the primary carcinoma there is cytoplasmic regression, nuclear shrinking, pyknosis and irregularity of stucture.

It is concluded that estrogens or dietheylstilbesterol produce hyperossification and that diethylstilbesterol with bilateral orchidectomy causes a regression of metastatic prostatic carcinoma plus a regression of the primary tumor.

M. G. W.

Prophylaxis of Upper Respiratory Infections in Children Treated with Oral Penicillin: J. H. Lapin, J. Pediat. *32*, 19-124 (1948).

A group of 148 children, ranging in age from 8 months to 10 years, were given 50,000 units of penecillin orally in buffered tablet form, daily, shortly before breakfast and shortly before supper, for 12 months. A control group of 110 children received no such medication. The two groups of children were equally distributed as to age and sex and lived in a congested metropolitan area. Before the study began, all had averaged 4 or more attacks per year of febrile upper respiratory infections including tonsillitis, pharyngitis, cervical adenitis, otitis media, bronchitis, etc. No penicillin levels were determined in this study but recent English and American work is quoted to show that this dosage will produce

bactericidal levels in children of these ages and weights. The control group showed no reduction in the average number of respiratory infections or in the average number of febrile days during the year under observation. The treated group, however, showed a reduction of 45% in the average number of upper respiratory infections from 5 during the preceding year to 2.3 in the year under study and a reduction of 25% in the number of febrile days from 16.7 to 4.2. No cases of the production of in vivo penicillin insensitive organisms occurred during this study.

H. A. P. M.

HUMAN BRUCELLOSIS—ITS SPECIFIC TREATMENT WITH A COMBINATION OF STREPTOMYCIN AND SULFADIAZINE: W. W. Spink, W. H. Hall, J. M. Shaffer, A. I. Braude, J. Amer. Med. Assn. *136*, 382-387 (1948).

DURING the past ten years, specific antribrucella therapy has been investigated at the University of Minnesota Hospitals, experimentally and in 35 sporadic cases of human brucellosis which were proven bacteriologically.

A chick embryo technic was utilized for the screening of therapeutic agents. The combined administration of sulfadiazine and streptomycin proved to be more effective than either agent alone by this method. A remarkable correlation of therapeutic results in chick embryos and human patients was observed.

Nine patients have been treated with a combination of sulfadiazine and streptomycin, which has yielded more satisfactory results than any other therapy used to date. It is recommended for the present that streptomycin be administered intramuscularly in doses of 0.5 Gm. every 6 hours for 7 days. Therapy with sulfadiazine should be initiated simultaneously with an oral dose of 4.0 Gms. and then 1.0 Gm. every 4 hours thereafter for at least 2 and preferably 3 weeks.

It is to be emphasized that the results of the combined therapy reported here are based on treatment of patients having brucellosis proven bacteriologically. The criterion of diagnosis is essential when dealing with such a variable disease as brucellosis for research purposes. However, the authors also treated other patients having febrile illness with clinical features of brucellosis and high agglutinin titres for Brucella with good success. Whether such therapy will be effective for the so-called chronic brucellosis remains to be investigated.

It is possible that another sulfonamide or combination of sulfonamides might be more effective than sulfadiazine and that another antibiotic may be discovered which is more effective than streptomycin.

W. G. B. C.

INDICATIONS FOR THYROIDECTOMY FOR HYPERTHYROIDISM. G. Crile, Jr., Western J. Surg. Obst. Gynec. *55*, 573-577 (1947).

ACCORDING to Crile, thiouracil has proven to be an effective means of preparing patients with severe hyperthyroidism for thyroidectomy. Its relatively high toxicity, however, makes it unsuited for general use as a definitive treatment. Propylthiouracil is relatively safe and though less effective, affords a good means

of controlling hyperthyroidism without undue danger of toxic effects. The author used 200-300 mg. daily of prophylthiouracil in the treatment of 218 patients. This dosage was not always adequate to maintain a remission. In a number of patients who discontinued treatment of their own accord after a few months of therapy, there were frequent recurrences. If the patient is co-operative and can be observed at intervals by a physician who is familiar with the structure of the thyroid and the pharmacologic action of the antithyroid drugs, treatment can be extended over long periods. When the patient is unco-operative or cannot be observed periodically, it is preferable to perform a thyroidectomy. Patients with small or moderate sized diffuse goitres, with mild or moderate hyperthyroidism, patients with recurrent hyperthyroidism, and patients whose life expectancy is short, regardless of the type of goitre, may safely be given a trial on medical management for a year or more. If they respond favorably, and prefer medical treatment, this may be continued indefinitely. If they respond poorly, or desire surgery, a subtotal throidectomy should be performed.

W. G. B. C.

The Human Conceptus During the First Two Weeks of Gestation: J. Rock and A. T. Hertig: Amer. J. Obs. & Gynec. 55, 1-188 (1948).

As of June 1947 these workers had obtained 26 very young embryos from 104 uteri, removed for therapeutic reasons, from women who had demonstrated normal fecundity and in whom coitus had occurred during the ovulation phase. This suggested a pregnancy rate of only 25%. Of these same 26 ova, twelve were considered to be defective (47%) either in trophoblast, in embryo proper or in both and all of these might be aborted. These 26 fertilized ova with their associated endometrial tissue give strong indications that in both long and short menstrual cycles, ovulation occurs about the fourteenth day before the onset of menstruation.

Within 48 hrs. after ovulation the human ovum has entered the tube, is fertilized and has divided into two blastomeres. Probably fertilization must occur within twelve hrs. By the third day it has reached the uterine cavity and by the fourth day is in an unfixed morula stage. The morula acquires a cavity and about the sixth day makes contact with the uterine wall and cavity collapses. By the ninth day, the cavity has redistended and an embryo consisting of two layers is within the cavity, the amniotic cavity is beginning and the outer layer of trophoblast has spaces in it for maternal blood. By the twelfth day the embryo consists of three layers, the amniotic cavity is larger and there is anlage of yolk sac. By fourteenth day the inner layer of trophoblast with primitive mesoblast shows the beginnings of the fetal placental circulation and amniotic cavity has almost surrounded the fetus.

Thus by the time of the first missed menstrual period the conceptus, which measures about three millimeters in diameter is embedded in endometrium, amniotic cavity is completed, and there is ground plan for placental circulation and yolk sac.

E. J. G.

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National Research Council of Canada. **Assoc. Committee on Medical Research. Subcommittee on burns; Treatment of thermal burns,** ed. 2. rev. 1944.
Page, J. D.: **Abnormal psychology.** 1947.
Pijoan, M. and Yeager, C. H.: **A handbook of commonly used drugs.** 1947.
Potter, E. L.: **Rh . . . its relation to congenital hemolytic disease and to intragroup transfusion reactions.** 1947.
Pye, W.: **Surgical handicraft,** ed. 15. 1947.
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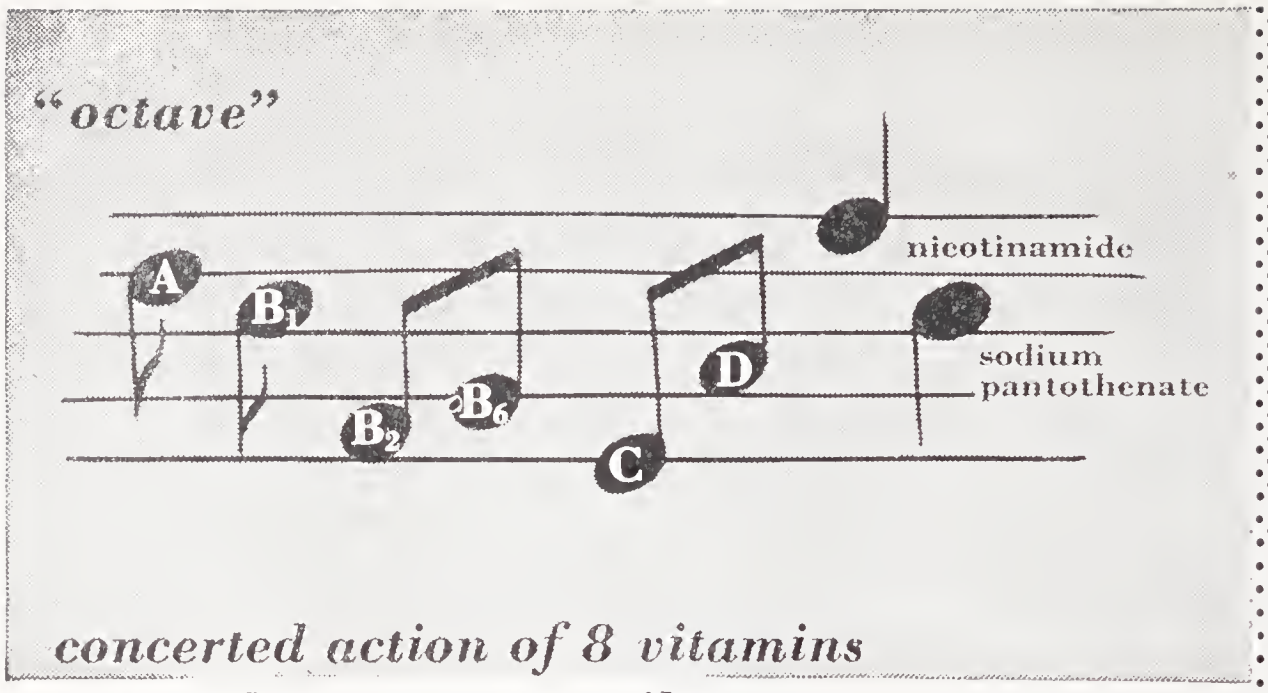

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REFERENCES:

1. *Proc. Soc. Exper. Biol. & Med. 55, 228, 1944.*
2. *Am. Heart Journ. 28, 759, 1944.*
3. *Act. Med. Scandin. 104, 527, 1940.*

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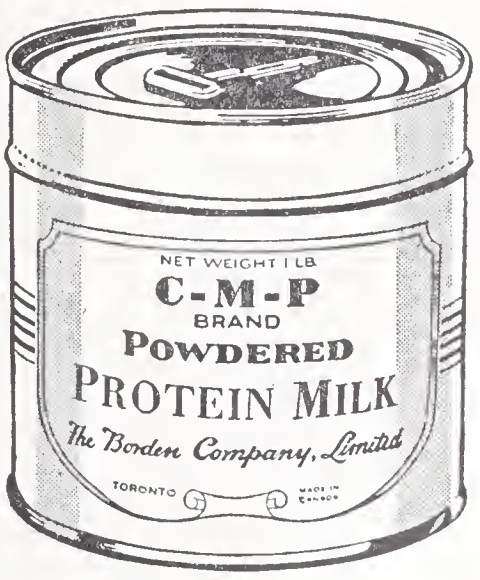

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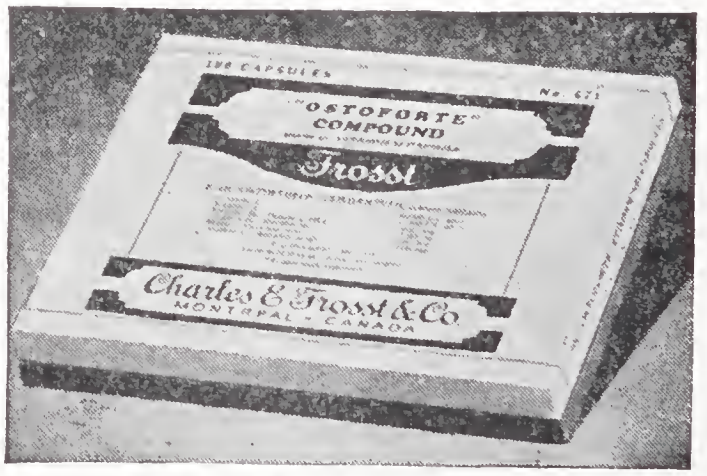

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